

FAK-tyrosine phosphorylation than the adjacent repeats III8, etc. The assumption is advanced that in the case of physiological concentrations of cellular fibronectins, the binding of the tetrapeptide RGDS (SEQ ID NO: 5) from III10 to the integrins possibly produces a signal of inadequate strength for the cell adhesion, so that no tyrosine-phosphorylation response arises from the interaction between III10 and integrin-mediated mechanisms. It is further assumed that the difference with respect to the response to the various mediated cell adhesions is produced by a varying activation of various small GTP-binding proteins. Three of these proteins -- cdc42, rac and rho -- which all are members of the ras-superfamily, play important roles in the case of cell-morphological changes. cdc42 acts sequentially upstream from rac and directly induces the appearance of filopodia (Nobes, C. D. and Hall, A., 1995, Rho, rac and cdc42 GTPases Regulate the Assembly of Multimolecular Focal Complexes Associated with Actin Stress Fibers, Lamellipodia and Filopodia, *Cell*, **81**, 53-62). The activation of rac is then responsible for the formation of lamellipodia and the network of actin filaments between the filopodia. Further downstream, rho can be activated by rac and induces focal adhesion and actin stress fibers. All of these events depend on the activation of tyrosine kinase, and it is assumed from FAK that it is involved in these processes. Chen and Culp make the conjecture that the morphological differences between cells that are adherent via 7-ED<sub>b</sub>-8 as well as cells that are adherent via 8-9-10 are based on the varying activation of the small GTP-binding proteins. The above suggests that an adhesion via 8-9-10 via the integrin-mediated signal path finally leads to an activation of rho to produce focal adhesions and actin stress fibers, while the adhesion of BALB/c-3T3 cells via 7-ED<sub>b</sub>-8 leads only to an activation of cdc42 proteins and rac proteins, but does not activate rho. For the above-mentioned speculations, however, data are presented in neither of the two studies.

Please delete the paragraph on page 20, lines 24 to 25, and replace it with the following paragraph:

Fig. 6 shows the partial sequences of synthetic peptides (SEQ ID NOS 4, 6-8, 1, 9-18, 2-3, 19-22, respectively in order of appearance) from the ED<sub>b</sub>-fibronectin domains used in Fig. 5;

Please delete the paragraph on page 28, lines 8 to 11, and replace it with the following paragraph:

**Fig. 6** shows the partial sequences of the synthetic ED-B peptides (SEQ ID NOS 4, 6-8, 1, 9-18, 2-3, 19-22, respectively in order of appearance) with the selected sequence designations that are removed from the total sequence of the ED<sub>b</sub>-fibronectin domains. The one-character code for amino acids is used.